Hypertensive-Ischemic Leg Ulcers

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SUMMARY

Ischemic ulcers of the leg having characteristics different from those of ordinary leg ulcers have been observed in a small number of hypertensive patients, mostly women, during the past few years.

Such ulcers are usually located above the ankle. They begin with a small area of purplish discoloration at the site of slight trauma, and progress to acutely tender ulceration.

In studies of tissue removed from the margin and the base of an ulcer of this kind, obliterative arteriolar sclerotic changes, ischemic-appearing connective tissue and inflammatory changes were noted.

Two additional cases are reported.

IN the course of several years the authors have observed a small number of patients who had painful, ischemic ulcers of the leg that differed from the usual ulcers of the extremities. The lesions occurred in almost every instance in women, and all the patients had a history of hypertensive disease of long duration.

Hines, who first noted the condition in a patient in 1941, published a preliminary report[4] in 1946 describing the clinical and histopathological features observed in 11 patients with ischemic ulcers occurring in the presence of hypertension. It was thought that the condition might be a new syndrome, and Hines postulated that changes similar to those in the retinal vessels of hypertensive patients were present in the arterioles of the skin and subcutaneous tissues, giving rise to superficial infarctions in the skin. Martorell[7] and Valls-Serra[8] at about the same time also reported studies of ischemic-appearing leg ulcers in hypertensive patients. Wright[9] reviewed these studies and added reports of cases observed by him.

Since the preliminary report in 1946, Hines and Farber[5] have observed 24 additional patients with hypertensive-ischemic leg ulcers. All but two of the patients were women.

DESCRIPTION OF A TYPICAL LESION

The ulcers are usually located on or above the lateral malleolus and range in size from 1 to 10 cm. in diameter. The border is soft and ill-defined and the base is ischemic-appearing. Usually the ulcer is superficial and the skin adjacent to it in most cases is normal in appearance. Cutaneous sclerosis is absent; hemosiderosis around the lesion is slight or absent.

DEVELOPMENT AND COURSE OF THE ULCER

Often there is history of slight trauma at the site of the lesion. The first sign is a superficial purplish discoloration, 0.5 to 1 cm. in diameter. This progresses by peripheral extension and the lesion breaks down centrally to form a superficial exquisitely tender ulcer. Healing is very slow and may not occur for four to six months. Rest in bed does not seem to influence the speed of recovery. Pressure dressings, supporting bandages and local antibiotic therapy are sometimes helpful. When healing does occur, the pain rapidly disappears and the only residual defect is a superficial, slightly depigmented scar.

PATHOLOGY

It is generally accepted that a diffuse disturbance of the arterial side of the vascular system exists in hypertension.[3,1,6] The cutaneous arterioles of patients with essential hypertension reveal sclerotic arteriolar changes identical to those found in the kidneys, retina, muscles and other organs of hypertensive patients.[2]

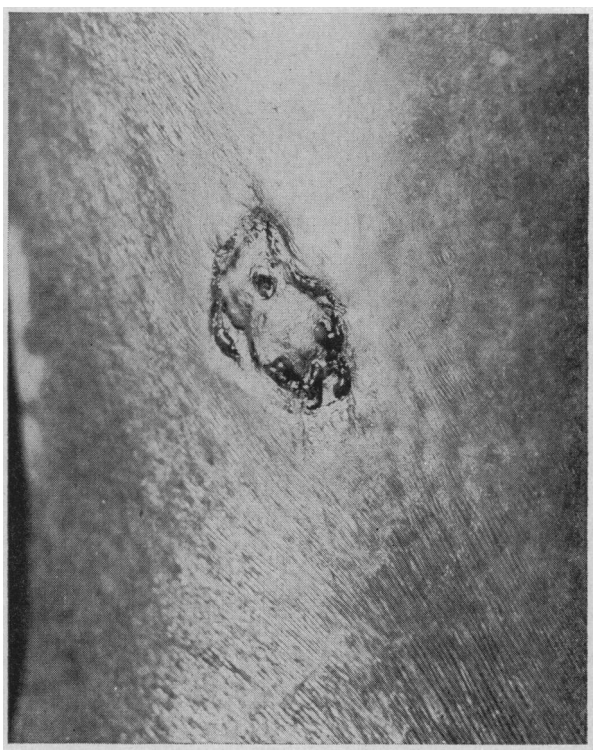

Figure 1.—Ischemic ulcer of the leg (Case 1).

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Biopsy specimens taken by excision and punch from the margin and from the base of an ulcer of the kind under discussion reveal arteriolar sclerotic changes. The arterioles are considerably thickened and many occluded. The cutis reveals some homogenization of the connective tissue. Many of the thickened and occluded arterioles are surrounded by a dense infiltration of polymorphonuclear leukocytes. Venules and veins were not thrombosed in the material studied. The sections studied serially revealed consistent obliterative sclerotic arteriolar changes, ischemic-appearing connective tissue, and inflammatory changes.

DIFFERENTIAL DIAGNOSIS

The lesion must be distinguished particularly from chronic pernio, from the ulcerations of livedo reticularis, from stasis ulcers due to chronic venous insufficiency, and from so-called "senile skin ulcers." Before a diagnosis of hypertensive ulcer of the leg is made, the usual causes of leg ulcers should be excluded. Careful examination of the arterial and venous systems should be made. The criteria for the diagnosis of hypertensive-ischemic leg ulcers should include: Hypertension; an ischemic-appearing ulcer unresponsive to conventional therapy; moderate to severe pain; indolence; typical changes of arteriolar sclerosis pathologically.

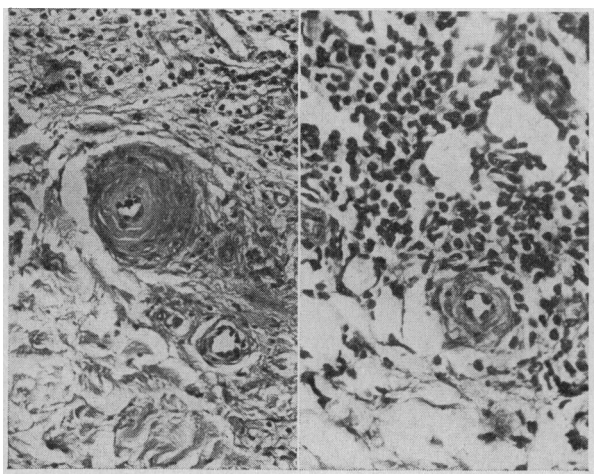

Figure 2.—(*Left*) Tissue from ulcer (Case 1). The media of the arteriole is hypertrophied and the lumen is narrowed. (*Right*) Tissue from ulcer (Case 1) showing a dense inflammatory reaction and a thickened arteriole in the mid-portion of the cutis.

In view of the fact that there is as yet little in the literature on this subject, two additional cases of ischemic ulcers of the leg in hypertensive patients are reported here.

CASE REPORTS

CASE 1: A 63-year-old Negro woman entered the Stanford University Hospitals on July 16, 1948, complaining of painful ulceration of the right leg. The lesion was reported to have started six months previously following slight trauma. It gradually enlarged and became increasingly painful. In January 1948 an ulcer had developed over the right Achilles tendon. This lesion was extremely painful but it healed after five months. The left leg had been amputated in 1927 because of severe third degree burns. There was no history of thrombophlebitis, syphilis, use of drugs or of blood dyscrasia.

Upon physical examination, a superficial, ischemic-appearing ulcer on the lateral aspect of the right leg was noted. The base of the ulcer contained indolent granulation tissue and the margin was soft. Slight pressure on the lesion caused exquisite pain. There was no evidence of varicose veins or of chronic venous insufficiency. Arterial pulsations were normal. The blood pressure was 230 mm. of mercury systolic and 120 mm. diastolic. Pronounced sclerosis and narrowing of the retinal arterioles were noted in the ocular fundi. The urine was normal, and results of serologic tests for syphilis were negative. The hemoglobin, erythrocyte and leukocyte determinations were within normal limits. There was no sickling of the erythrocytes. Cultures taken from the ulcer showed a heavy growth of coagulase-positive Staphylococcus aureus. Biopsy specimens were taken from the ulcer and from skin adjacent to it.

The lesion was treated by bed rest, topical applications of antibiotics and supporting bandages. The ulcer gradually became less painful and after seven months was completely healed.

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CASE 2: A 60-year-old white woman was first examined at the Stanford University Hospitals Nov. 23, 1948, because of an ulcer on the left ankle of two months' duration. The lesion was said to have begun at the site of a small bruise that had been received in a fall on a graveled road. It had enlarged peripherally and had broken open in the center to form a superficial ulcer which became so painful that walking was difficult. There was no history of phlebitis or of any serious illness. Intermittent claudication had never been noted. The blood pressure was known to have been elevated for ten years.

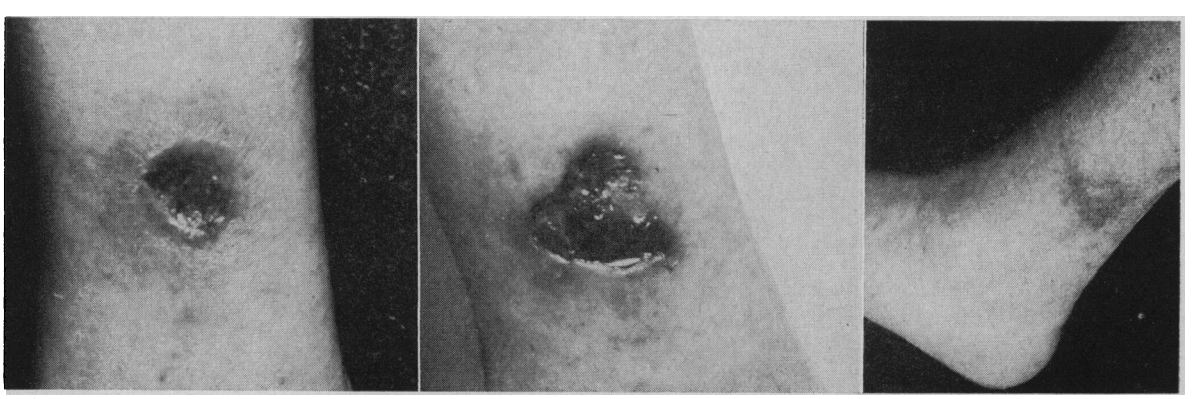

Figure 3.—(*Left*) Ischemic ulcer in the lateral surface of the leg (Case 2). (*Center*) Same ulcer two months later. (*Right*) Healed ulcer seven months after onset.

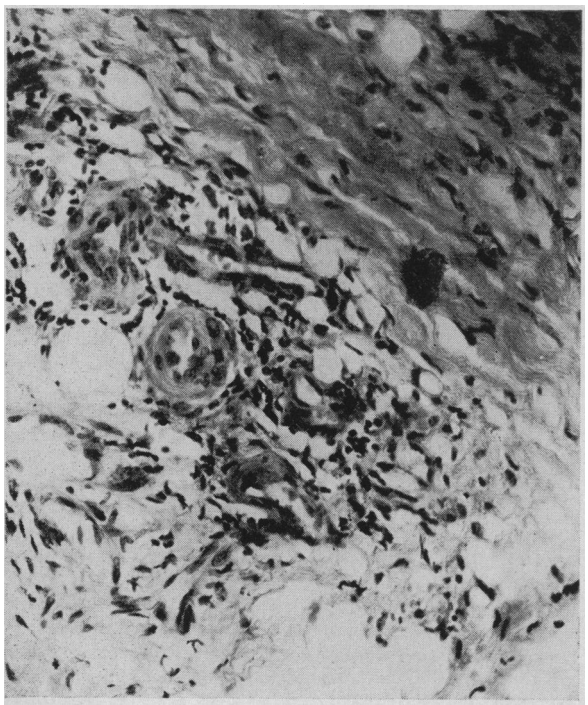

Figure 4.—Tissue from ulcer (Case 2) showing arteriolar sclerotic changes and perivascular inflammatory reaction.

On physical examination, a superficial, sharply punched-out, soft-edged ulcer, 1.5 cm. in diameter, was noted above the left lateral malleolus. No cutaneous sclerosis was present and only a small amount of pigmentation was scattered around the ulcer. Slight pressure on the lesion resulted in severe pain. The greater saphenous veins were dilated bilaterally but were apparently competent. Palpation of the peripheral vessels revealed slightly diminished pulsations of the dorsalis pedis arteries. There was no pallor following elevation of the feet and no rubor following dependency. A roentgenogram of the lower extremities showed no evidence of calcification of the peripheral vessels.

The blood pressure was 250 mm. of mercury systolic and 140 mm. diastolic. Results of laboratory studies of the blood and urine were within normal limits, and serologic tests were negative for syphilis. A culture from the exudate yielded a small growth of E. coli. A biopsy specimen was taken from the margin of the ulcer.

The patient was confined to bed and during the next two months the ulcer gradually enlarged, almost doubling in diameter. Following continued applications of various antibiotics, the ulcer began to heal and the pain diminished. Approximately seven months after the ulcer first appeared, healing was complete.

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Discussion by N. E. FREEMAN, M.D., *San Francisco*

Dr. Farber's presentation serves further to emphasize the importance of hypertensive ischemic ulcers of the leg.

Ulcer of the leg is not a fatal disease, but it may be incapacitating; and the loss of time, of both patient and physician, represents a serious economic burden. The very fact that leg ulcers are so frequently encountered has militated against careful study of them.

I am not prepared to accept the term "hypertensive ischemic leg ulcers" as an etiological diagnosis, chiefly because I do not feel that hypertension itself is anything more than a sign or a symptom of an underlying cardiovascular condition. But possibly this is just quibbling. The advantage of appending a name to some clinical condition rests in the fact that cases which fit into a certain category can be set aside for more intensive study. The disadvantage, however, is that as soon as a condition has been given a name it is thought to be understood, and the physician's interest may be stultified in that he can say to himself, "Oh, yes, this is a hypertensive ischemic leg ulcer."

There is just one question I should like to ask: Why do these ulcers appear only in the lower extremities? The circulation of blood is dependent upon the establishment of a gradient of pressure from the arterial to the venous side of the capillary network. Manifestly, in these patients whom Dr. Farber has described there is evidence of structural impairment of arterial supply. However, I believe more careful search should be made for evidences of impairment of venous return. Even though the patients gave no history of thrombophlebitis, had no varicose veins, and had little or no edema, I do not believe that the question of impairment of venous return has been ruled out. Careful studies by phlebography have often demonstrated the presence of deep venous occlusion or incompetent valves of the communicating veins of the legs in patients who have given no history of thrombophlebitis. Bauer of Sweden has demonstrated by venography how often even mild trauma to the lower extremities results in venous thrombosis. This venous thrombosis may be entirely unrecognized, both by the patient and by the physician, and yet, years later, may be followed by post-thrombotic sequelae.

Another point of interest is the degree of vasoconstriction which may for years have preceded the development of these leg ulcers. It is well known that prolonged vasoconstriction, as in Raynaud's disease, ultimately will lead to organic arterial obliteration of the smaller blood vessels. The ulcers of the leg in patients with livedo reticularis demonstrate, on histological study, lesions involving both the arteries and veins not unlike those which Dr. Farber has described.